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EXPERIMENTAL STUDY OF REFLEX SHOCK

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EXPERIMENTAL STUDY OF REFLEX SHOCK

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By

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I. EXPERIMENTAL STUDY OF PLEURAL SHOCK

The first description of pleural shock was given by ROGER in 1864, who attributed it to a reflex action from the stimulation of nerve-endings in the pleura. Since the artificial pneumothorax was introduced by FORLANINI in 1890 and the thoracic surgery has become a common practice, the number of pleural accidents is gradually increasing.

According to CAPPS, in any operative intervention on the pleural cavity, whether it is a therapeutic pneumothorax, a draining of empyema, a withdrawal of fluid by means of an aspirating needle or merely an exploratory thoracentesis, there lurks the danger of pleural shock. In this state a patient becomes faint, occasionally unconscious and pulse becomes weaker and weaker until it can no longer be detected by the finger. Convulsions and death are rare.

TAKEUCHI (1951) reported that by injecting tincture of iodine into the pleural cavity of rabbits he produced pleural shock, in which blood pressure fell, pulse rate became slower and pupils dilated. There were some evidences suggesting that pleural shock might be related to the vagus nerve.

Some time ago I experienced an accident that aroused my interest in this subject. A man, aged 27, underwent the aspiration of a pleural effusion. He suddenly became faint and then immediately lost consciousness, with the pupils dilated and the pulse almost impalpable and apparently slower. Routine emergency measures were done with no effect; then atropine was injected hypodermically, and the patient recovered in ten minutes. The essential feature in this case is obviously pleural shock caused by the puncture of the pleura. It seemed quite probable that the shock might be related to the vagus nerve, as the patient recovered with atropine. Suggested by this case, the following experimental study has been performed in cats.

EXPERIMENTAL TECHNIC

Cats, about 2 kg. in weight were used.

The blood pressure was recorded graphically by inserting a cannula into the right common carotid artery or the femoral artery and in order to measure the pulse rate exactly, an electrocardiograph was employed in some cases. Various stimulants were introduced into the pleural cavity with manometric control. At

necropsy, it was assured whether they had been injected exactly into the pleural cavity or not.

RESULTS

Series I: Thermal Stimulation of Pleura.

A) Injection of hot water 80°C. (5 cc) into the pleural cavity.

Table I. (A)

No.		Before injection	After injection				
			immediately	1′	3′	5′	10′
1	Blood pressure	120	120	114	114	110	
	Pulse rate	208	208	236	220	232	
2	Blood pressure	140	138	138	136	134	126
	Pulse rate	200		212	208	204	200

The blood pressure showed no marked drop and the pulse rate became rather frequent.

B) Injection of cold water 5°C. (5 cc) into the pleural cavity.

Table I. (B)

No.		Before injection	After injection				
			immediately	1′	3′	5′	10′
3	Blood pressure	160	180	164	164	170	174
	Pulse rate	240		200	228	228	180

The blood pressure raised a little, but the pulse rate decreased soon after injection.

Comment.

It was expected that pleural shock might result, if the pleura was stimulated by hot or cold water. However, there occurred no marked fall of blood pressure and no slowness of pulse rate referable to a neurogenic reflex. Namely, pleural shock could not be produced by the application of thermal stimulus to pleura.

Series II: The Chemical Stimulation of Pleura.

A) Injection of 5 % tincture of iodine (5 cc) into the pleural cavity.

Table II. (A)

No.		Before injection	After injection					
			immediately	1′	3′	5′	10′	15′
4	Blood pressure	200	230	176	164	160	160	160
	Pulse rate	180			160	140	148	136
5	Blood pressure	140	180	104	76	68	62	58
	Pulse rate	220	200	180	165	160	150	144
6	Blood pressure	124	111	96	90	64	Died 9′ later	
	Pulse rate	200		180	160	140		

7	Blood pressure	140	138	120	104	114	116	
	Pulse rate	200		190	180	180	200	

Table II. (B)

No.		Before injection	After injection							
			immediately	1′	2′	3′	4′	5′	10′	15′
8	Blood pressure	130	156	86	64	44	10	Died 5′ later		
	Pulse rate	166	160	128	88	52	32			
9	Blood pressure	144	190	96	100	108	104	108	130	140
	Pulse rate	220	176	176	172	172	168	168	192	208
10	Blood pressure	98	100	40	44	14	Died 4′ later			
	Pulse rate	224		72	64					

In all cases, there was a tendency to low blood pressure and bradycardia. In one case (No. 6), blood pressure fell about 28 mm Hg. a few minutes after injection and the animal died after about 9 minutes.

B) Injection of 10% hydrochloric acid (2 cc) into the pleural cavity.

These experiments were performed in May 1951, and in all animals without exception blood pressure fell and pulse rate became slower. In two animals, blood pressure fell about 50 mm Hg. immediately after injection and they died within five minutes.

Comment.

CORDIER (1910) injected iodine into the pleural cavity and produced pleural shock. CROIZER (1927) found that pleural shock

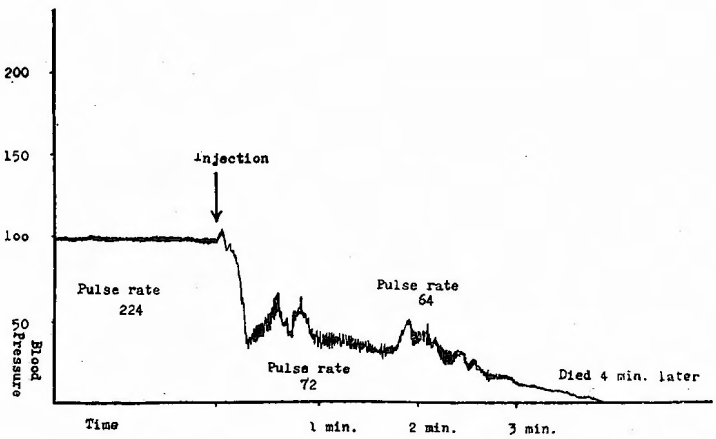

Fig. 1. Injection of 10% hydrochloric acid 2 cc into the pleural cavity. (No. 10)

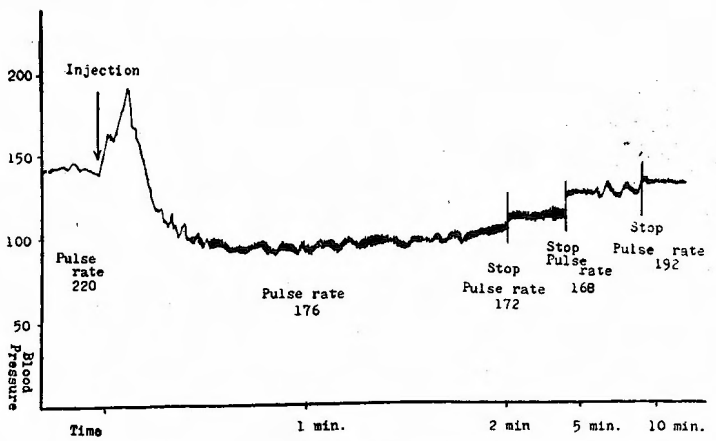

Fig. 2. Injection of 10% hydrochloric acid 2 cc into the pleural cavity. (No. 9)

could be induced when iodine was injected directly into the lung. In rabbits, TAKEUCHI (1951) produced pleural shock by injecting 5% tincture of iodine into the pleural cavity.

In my experiments, the injection of 5% tincture of iodine and 10% hydrochloric acid into the pleural cavity of cats always produced pleural shock, in which blood pressure fell and pulse rate became slower. It was confirmed at necropsy that iodine or hydrochloric acid had been exactly injected into the pleural cavity, the pleura showing discolouration as a result of strong stimulation.

Series III: Pharmacostimulation of the pleura.

A) Injection of 0.1% adrenaline (0.5 cc) into the pleural cavity.

Table III. (A)

No.		Before injection	After injection					
			immediately	1′	2′	3′	4′	5′
11	Blood pressure	150	190	140	150	160	170	170
	Pulse rate	152		124	124	124	124	124
12	Blood pressure	164	150	136	136	136	136	136
	Pulse rate	220		260	264	260	260	260

B) Injection of 0.1% pilocarpine solution (1 cc) into the pleural cavity.

Table III. (B)

No.		Before injection	After injection					
			immediately	1′	2′	3′	4′	5′
13	Blood pressure	130	150	120	126	134	134	140
	Pulse rate	216			196			
14	Blood pressure	160	170	148	150	148	148	146

The blood pressure fell gradually and pulse rate somewhat decreased, but it seemed uncertain that the changes were due to a neurogenic reflex.

C) Injection of acetylcholine (10 mg) into the pleural cavity.

Table III. (C)

No.		Before injection	After injection					
			immediately	1′	2′	3′	4′	5′
15	Blood pressure	140	160	46	40	66	80	82
	Pulse rate	172						120
16	Blood pressure	136	130	24	50	54	60	62
	Pulse rate	200				124		

As soon as acetylcholine was injected into the pleural cavity, blood pressure fell rapidly and pulse rate decreased pronouncedly.

Comment.

By the injection of adrenaline and pilocarpine, reflex shock was not evident. By acetylcholine, however, there occurred marked fall of the blood pressure and bradycardia, which seemed likely due to a neurogenic reflex. This is a finding similar to that following the injection of tincture of iodine or hydrochloric acid, but is more pronounced than the latter, although the fall of blood pressure and bradycardia in case of acetylcholine may be the result of its absorption into general circulation.

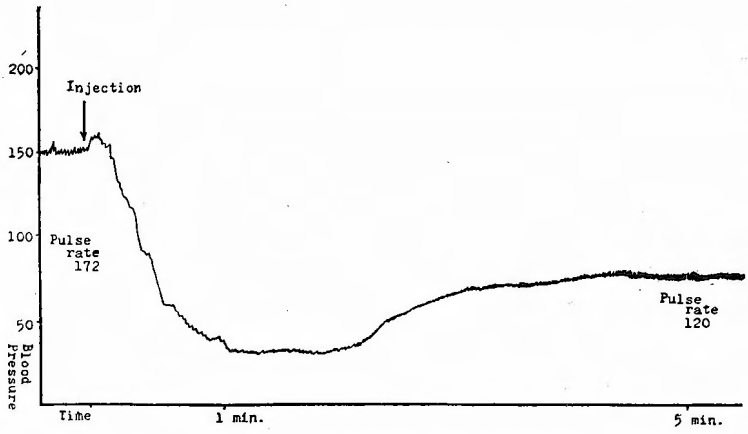

Fig. 3. Injection of acetylcholine 10 mg into the pleural cavity. (No. 15)

Series IV : Injection of Various Chemicals into the Pleural Cavity after Preliminary Injection of Atropine.

In the preceding experiments, it was demonstrated that the syndrome of pleural shock is almost the same as that induced by the intrapleural or hypodermic injection of acetylcholine, namely low blood pressure and bradycardia. It may be expected that preliminary administration of atropine will prevent the pleural shock following the injection of various chemicals into the pleural cavity.

Cats were given 0.05% atropine 0.02 cc (0.01 mg) per kilogram of body weight subcutaneously twice at an interval of twenty minutes. Then various chemicals were injected into the pleural cavity.

A) Injection of 5% tincture of iodine (5 cc) into the pleural cavity after preliminary injection of atropine.

Table IV. (A)

No.		Before injection	After injection					
			immediately	1′	3′	5′	10′	15′
17	Blood pressure	160	170	164	162	160	160	164
	Pulse rate	186	180	192	204	204	192	204
18	Blood pressure	170	220	180	180	164	156	160
	Pulse rate	172	200	192	184	180	184	184

At the moment when tincture of iodine was injected into the pleural cavity, cats showed manifestations of pain, and the blood pressure temporarily increased, but soon returned to the previous level. Pulse rate increased. Thus pleural shock, which was otherwise expected to occur, failed entirely to occur by the premedication of atropine.

B) Injection of 10% hydrochloric acid (2 cc) into the pleural cavity after preliminary injection of atropine.

Table IV. (B)

No.		Before injection	After injection					
			immediately	1′	3′	5′	10′	15′
19	Blood pressure	100	220	106	100	100	104	104
	Pulse rate	184	184	176	160	174	180	200
20	Blood pressure	144	220	140	140	134	136	140
	Pulse rate	208	192	192	188	180	176	200

pulse rate became a little slower, but blood pressure did not drop. Pleural shock was prevented by the premedication of atropine.

C) Injection of acetylcholine (10 mg) into the pleural cavity after preliminary injection of atropine.

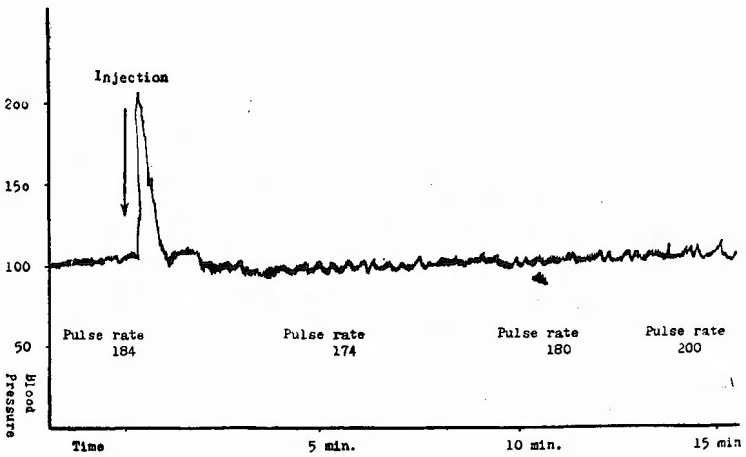

Fig. 4. Injection of 10% hydrochloric acid 2 cc into the pleural cavity after preliminary injection of atropine. (No. 19)

Blood pressure fell after injection, presumably due to the direct action of absorbed acetylcholine to the heart. Pulse rate did not become slower.

Table IV. (C)

No.		Before injection	After injection				
			immediately	1′	2′	3′	4′
21	Blood pressure	100	102	90	80	82	
	Pulse rate	210			192		
22	Blood pressure	112	114	70	71	84	86
	Pulse rate	224			220		

Comment.

For the development of a pleural shock, one should think of vagal reflex. In this experiment, therefore, I administered atropine subcutaneously before the injection of various chemicals into the pleural cavity. The result was that pleural shock was arrested by atropine. I believe this is a direct evidence for the main

rôle of the vagus-nerve in the development of pleural shock.

Series V: Influence of Season and Atmospheric Temperature upon the Experimental Occurrence of Pleural Shock.

A) Injection of 10% hydrochloric acid (2 cc) into the pleural cavity in about 30°C atmospheric temperature.

This experiment was carried out in July 1951. The rectal temperature of cats was abouf 38°C.

Table V. (A)

No.		Before injection	After injection						
			immediately	1′	3′	5′	7′	10′	15′
23	Blood pressure	134	184	122	124	106	100	108	
	Pulse rate	236	126	170	150	178	178	160	
24	Blood pressure	140	190	130	142	140	130	130	
	Pulse rate	188	160	216	192	180	184	188	
25	Blood pressure	126	190	110	114	116	116	120	138
	Pulse rate	224	140	172	180	188	212	224	228
26	Blood pressure	108	180	114	124	110	80	78	
	Pulse rate	212	200	188	192	224	228	224	
27	Blood pressure	156	220	164	162	144	138	140	138
	Pulse rate	248		228	240	232	200	192	180
28	Blood pressure	160	220	164	104	86		94	104
	Pulse rate	208	148	104	200 (arhythmia)			120	176

The typical pleural shock, in which blood pressure fell remarkably and pulse rate became slower, was observed only in No. 28. However there was a suggestive pleural shock in animals Nos. 23, 25 and 26. The others did not show any sign of reflex shock.

B) Injection of 10% hydrochloric acid (2 cc) into the pleural cavity after cooling the body with ice about 30° C atmospheric temperaure.

Table V. (B)

No.		Before injection	After injection						
			immediately	1′	3′	5′	7′	10′	15′
29	Blood pressure	142	206	116	114	120	114	120	120
	Pulse rate	180	180	148	128	128	132	168	180
30	Blood pressure	150	200	180	22	Died 4′ later			
	Pulse rate	224		144	48				
31	Blood pressure	132	184	90	72	74	46	Died 9′ later	
	Pulse rate	184		52	60	60	68		

32	Blood pressure	130	172	74	62	62	62	74	90
	Pulse rate	180	108	136	112	180	180	128	144
33	Blood pressure	106	190	88	76	84	90	80	92
	Pulse rate	216	168	172	176	176	172	160	172

Cats were cooled until the rectal temperature fell to about 35.5°C, and one hour afterward, hydrochloric acid was injected into the pleural cavity.

Fall of blood pressure and bradycardia were observed in nearly all cases. In No. 30, which died after about 4 minutes, blood pressure dropped alarmingly. Comment.

From the fact that pleural shock was difficult to take place in summer, it was supposed that there might be some relation between the season or the atmospheric temperature and the pleural shock. Experiment Series V. (B) demonstrated that the cooling of the body was a predisposing factor of pleural shock. According to ISENSCHMIDT, descending of body temperature gives rise to the irritative state of the parasympathetic nervous system. This may explain the result in this experiment.

Series VI: Influence of the Section of the Bilateral Vagal Nerves upon Pleural Shock.

By cutting the vagal nerves on both sides in the neck, cats become dyspnoeic, but sooner or later recover with artificial respiration in most cases. After recovery to the normal condition, the following experiments were performed.

A) Injection of 10% hydrochloric acid (2 cc) into the pleural cavity after the section of the bilateral vagal nerves in about 30°C atmospheric temperature.

Table VI. (A)

No.		Before injection	After injection					
			immediately	1′	3′	5′	7′	10′	15′
34	Blood pressure	80	140	70	56	68	68	68	80
	Pulse rate	232	216	228	168	172	172	180	180
35	Blood pressure	90	120	74	54	64	56	74	
	Pulse rate	264		272	236	264	248	240	
36	Blood pressure	116	200	94	86	90	90	90	100
	Pulse rate	168		144	120	128	132	136	152
37	Blood pressure	144	220	140	128	102	90	90	96
	Pulse rate	176	160	164	164	160	164	156	160
38	Blood pressure	180	250	200	196	180	174	150	
	Pulse rate	220	248	232	208	212	208	208	

The typical pleural shock appeared in no case, although a tendency to shock was noticed in Nos. 34 and 36.

In view of the fact that the typical pleural shock did not occur also in Series

V. (A). There is no much significance in the result of this experiment, that the pleural shock was arrested by the section of the vagi in summer. The most one can say, may be that there is no appreciable difference between Series V. (A) and Series VI. (A).

B) Injection of 10% hydrochloric acid (2 cc) into the pleural cavity after cooling the body together with section of the bilateral vagal nerves in about 30°C atmospheric temperature.

Table VI. (B)

No.		Before injection	After injection						
			immed-iately	1′	3′	5′	7′	10′	15′
39	Blood pressure	144	146	142	138	140	140	140	138
	Pulse rate	200	196	196	200	208	188	188	192
40	Blood pressure	106	144	116	116	104	108	108	106
	Pulse rate	152		160	172	172	192	168	168
41	Blood pressure	134	150	104	108	108			
	Pulse rate	140		156	180	180			
42	Blood pressure	156	146	80	Died 3′ later				
	Pulse rate	192		184					
43	Blood pressure	156	164	150	150	150	150	150	146
	Pulse rate	216		224	232	216	216	224	228

In only one case (No. 42) death occurred 3 minutes after injection of hydrochloric acid, but in others, fall in blood pressure and bradycardia were not produced.

Comment.

For the reason that fall in blood pressure and bradycardia did not take place after the section of vagi and the preliminary freezing, it is decidedly clear that the pleural shock is arrested by the section of the bilateral vagal nerves. In short, the vagus nerve plays a principal rôle in the development of pleural shock.

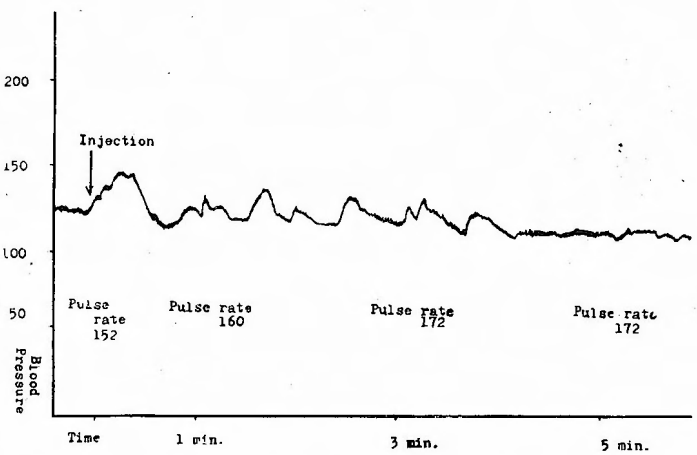

Fig. 5. Injection of 10% hydrochloric acid 2 cc into the pleural cavity after section of bilateral vagal nerves (atmospheric temperature about 30°C). (No. 40)

SUMMARY

(1) The pleural shock did not result from the injection of hot or cold water into the pleural cavity.

(2) It developed by the injection of iodine or hydrochloric acid into the pleural cavity. In this state, there were low blood pressure and bradycardia.

(3) Similarly it developed by the injection of acetylcholine into the pleural cavity, but not by adrenaline or pilocarpine.

(4) The pleural shock was arrested by the subcutaneous administration of atropine before injection of various shock-producing chemicals into the pleural cavity.

(5) The freezing of the body makes a predisposing state of pleural shock.

(6) After cutting the both vagi, pleural shock did not occur. Therefore, pleural shock in cats seems to appear by a reflex through the vagus nerve.

(7) The typical manifestations of pleural shock, i. e. low blood pressure and bradycardia are analogous to those of the initial shock after head injuries, as Prof. Araki stated.

II. EXPERIMENTAL STUDY OF REFLEX SHOCK IN ABDOMEN

The primary shock is believed to be a cardiovascular reflex phenomenon appearing immediately after injury, but the symptomatology is not well established. It seems to consist of low blood pressure, slow pulse, cold skin, pallow face, loss of consciousness and perspiration, whereas in the secondary shock pulse rate becomes frequent and blood pressure falls, as a result of progressive reduction in circulating blood volume. Almost the same features as in the primary shock appear in the case of fainting spells due to cerebral anemia following trivial stimuli, and initial shock after head injuries

GOLTZ observed that on repeated strokes on the frog's abdomen the heart beats become slower or even cease, and he attributed it to reflex inhibition of the heart through the vagus nerve. It has been described that the circulatory disturbance of the primary shock type are occasionally experienced in abdominal diseases accompanied by severe pain, for instance, Japanese "ATEMI", acute pancreatic necrosis, perforation of a gastric ulcer or cancer, and acute intestinal strangulation.

To clarify whether a primary shock with bradycardia and low blood pressure may be induced experimentally from the abdomen, especially from the upper abdomen, the present experiments in cats have been performed.

EXPERIMENTAL TECHNIC

The method of experiments is the same as that in the pleural shock. Anesthesia was not done in all animals.

RESULTS

Series I: Chemical Stimulation of Abdominal Cavity.

In case of acute perforation of the stomach or intestine, pulse is usually feeble and frequent. However, bradycardia and low blood pressure are occasionally seen, especially immediately after the perforation, presumably as the results of intra-abdominal vagal stimulation. To reveal whether the latter type of shock could be induced by the injection of stimulant chemicals into abdomen, the following experiments were performed.

1) Injection of 5% tincture of iodine 10cc into the abdominal cavity.

A) Injection into the upper abdominal cavity.

Table VII. (A)

No.		Before injection	After injection					Remarks
			immediately	3′	5′	7′	10′	
44	Blood pressure	150	200		152		144	
	Pulse rate	180	210		200		200	
45	Blood pressure	186	260	170	160	160	158	
	Pulse rate	200	200	200	200	200	208	
46	Blood pressure	160	210	148	140	140	130	
	Pulse rate	220	220	220	220	200	192	
47	Blood pressure	150	176	140	130	130	100	Died 30′
	Pulse rate	240	240	232	220	208	120	later

B) Injection into the lower abdominal cavity.

Table VII. (B)

No.		Before injection	After injection				
			immediately	3′	5′	7′	10′
48	Blood pressure	170	184	164	164	160	160
	Pulse rate	168	200	200	200	220	220

2) Injection of hydrochloric acid into the upper abdominal cavity.

A) Injection of 10% hydrochloric acid 10 cc.

Table VIII. (A)

No.		Before injection	After injection				
			immediately	3′	5′	7′	10′
49	Blood pressure	120	186	110	100	100	104
	Pulse rate	184		220	200	192	188

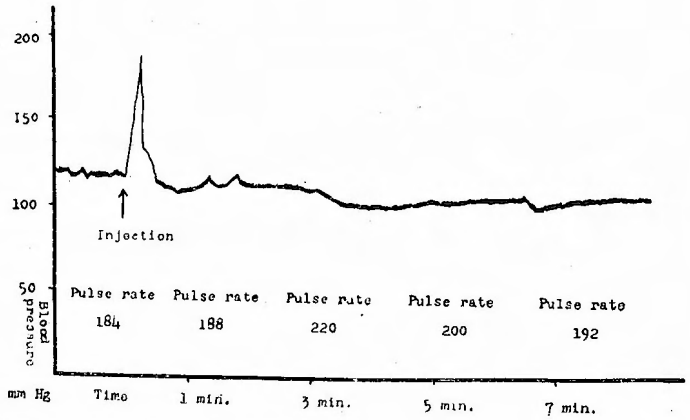

Fig. 6. Injection of 10% hydrochloric acid 10 cc into the upper abdominal cavity. (No. 49)

B) Injection of 20% hydrochloric acid 7.5 cc.

Table VIII. (B)

No.		Before injection	After injection				
			immediately	3′	5′	7′	10′
50	Blood pressure	130	210	110	74	68	44
	Pulse rate	208	224	278	250		232

C) Injection of 30% hydrochloric acid 2.0 cc.

Table VIII. (C)

No.		Before injection	After injection				
			immediately	3′	5′	7′	10′
51	Blood pressure	120	190	102	90	80	46
	Pulse rate	248		248	256		256

D) Injection of 50% hydrochloric acid 10 cc.

Table VIII. (D)

No.		Before injection	After injection			Remarks
			immediately	3′	5′	
52	Blood pressure	110	182	60	38	Died 7′later
	Pulse rate	172	204	228	200	

Comment.

In all these experiments, blood pressure and pulse rate increased immediately following injection probably due to a severe pain, and then gradually fell.

WEGNER (1876) stated that the main cause of death in acute peritonitis was the absorption of toxic substances produced in the abdominal cavity. Similarly, the death of animals Nos. 47 and 52 might be caused by the absorption of iodine or hydrochloric acid. There was no evidence of reflex shock.

Series II: Injection of 10% Hydrochloric Acid 5 cc into the Abdominal Cavity of the Animals with Dysfunction of the Liver.

FINE (1949) and SHORR (1950) report that the hepatic anoxia is an important change in the shock-like state. It has been known that a major operation on a patient with hepatic dysfunction is liable to produce secondary shock. Therefore, it was expected that a primary shock might be more easily produced, when the function of the liver was disturbed. Carbon tetrachloride was given or the common bile duct was ligated, in order to impair the hepatic function of cats irreversibly.

1) The deterioration of the liver by carbon tetrachloride.

Cats had been given 20% carbon tetrachloride olive oil solution 1 cc per kilogram of body weight subcutaneously from 3 to 5 times every other day. The following experiments were carried out two days after the final injection.

A) Injection of 10% hydrochloric acid 5 cc into the abdominal cavity after

treatment with carbon tetrachloride 3 times every other day.

Table IX. (A)

No.		Before injection	After injection				
			immediately	3′	5′	7′	10′
53	Blood pressure	114	170	110	110	114	114
	Pulse rate	200		220	212	210	210

B) The same injection after treatment with carbon tetrachloride 4 times every other day.

Table IX. (B)

No.		Before injection	After injection				
			immediately	3′	5′	7′	10′
54	Blood pressure	90	146	80	74	72	72
	Pulse rate	232	240	240	236	236	242
55	Blood pressure	112	168	106	100	94	94
	Pulse rate	200	228	228	240	228	228

2) The disturbance of hepatic function due to ligation of the common bile duct.

The cats did not eat anything after the ligation of common bile duct. 3 to 5 days later the liver showed spotted surface and fat-degeneration. Subcutaneous fat-tissues exhibited icteric colour. Injection of hydrochloric acid into the abdominal cavity was performed 3 resp. 5 days following the ligation of common bile duct.

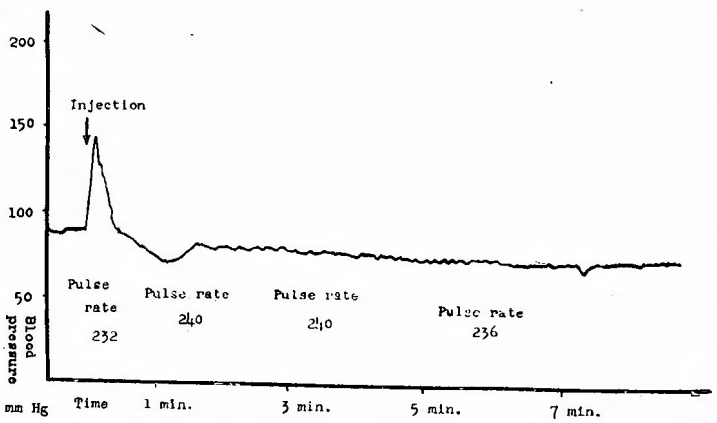

Fig. 7. Injection of 10% hydrochloric acid 5 cc into the upper abdominal cavity of cats, treated with carbon tetrachloride 4 times every other day. (No. 54)

Table X.

No.		Before injection	After injection							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
56	Blood pressure	130	210	116	84	70	86	94	104	104
	Pulse rate	148		196	188	164	140	128		152
57	Blood pressure	130	164	132	130	130	130	130	130	130
	Pulse rate	200		232	228	228	224	224	224	224

Comment.

In every case, blood pressure rose a little immediately after injection of hydrochlroic acid and soon fell. However, pulse rate somewhat increased.

In short, the primary shock showing bradycardia and low blood pressure did not occur even in the cases of irreversible damage of the liver.

Series III : Injection of 10% Hydrochloric Acid 5 cc into the Upper Abdominal Cavity in Splanchnectomized Cats.

It is known that cerebral anemia with bradycardia and lower blood pressure is liable to occur in vagotonics, and that the electric stimulation in the proximal portion of cervical vagus nerve exerts an inhibitory action on the heart, resulting in low blood pressure. Therefore, it should be expected that after cutting sympathetic nerves on both sides, the parasympathetic nerves in the abdomen become predominant, and that reflex phenomenon through the parasympathetic nerve would be more easily produced. Thus injection of hydrochloric acid into the abdominal cavity was performed about one week after the section of upper lumbar sympathetic chains on both sides.

Table XI

No.		Before injection	After injection							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
58	Blood pressure	100	151	96	84	86	81	80	76	76
	Pulse rate	184	128	160	200	200	196	196	192	192
59	Blood pressure	150	200	160	150	146	146	144	144	144
	Pulse rate	160	128	144	148	148	148	148	150	150
60	Blood pressure	120	190	120	121	110	110	106	106	106
	Pulse rate	212		208	228	200	188	188	188	188

Comment.

In every case blood pressure fell gradually but remarkably, and pulse rate became slower immediately following injection and gradually increased later. In Nos. 58 and 59, the blood pressure fell temporarily and pulse rate decreased about one minute after injection, but soon returned to the former level. This response was supposed to be a vagal reflex, but not so strong as to be called a primary shock.

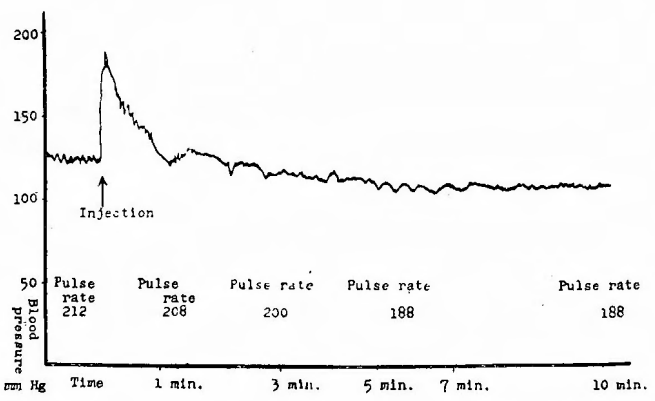

Fig. 8. Injection of 10% hydrochloric acid 5 cc into the upper abdominal cavity of splanchnectomized cat. (No.60)

Series IV : Injection of 10% Hydrochloric Acid 5 cc into the Upper Abdominal

Cavity after Fasting.

POPOW (1885), HAYASHI (1924), and TABUCHI (1943) observed that the hepatic function is impaired in the state of starvation. SUZUKI and his associates (1951) reported that the secondary shock due to a nervous stimulation is liable to occur in rabbits fasting for 3 to 5 days. For a long time, it has been believed that starvation often produces shock-like symptoms, i. e., starvation is a predisposing state of secondary shock. Thus the following experiments were performed in cats, whose liver had been reversibly damaged by fasting.

1) Injection of 10% hydrochloric acid 5 cc into the upper abdominal cavity of cats fasting for 2 days.

Table XII.

No.		Before injection	After injection						Remarks
			immed-iately	1′	2′	3′	4′	5′	
61	Blood pressure	76	84	54	44	50	40	34	Died 6′ later.
	Pulse rate	220		80	80	80	100	100	

2) The same injection in cats fasting for 3 days.

Table XIII

No.		Before injection	After injection							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
62	Blood pressure	110	156	82	120	80	80	80	74	60
	Pulse rate	180		92	160	200	200	200	192	180
63	Blood pressure	110	172	110	104	104	100	102	98	96
	Pulse rate	220		228	200	192	180	184	200	208
64	Blood pressure	130	200	144	106	96	94	94	92	100
	Pulse rate	168		132	148	162	172	176	176	172

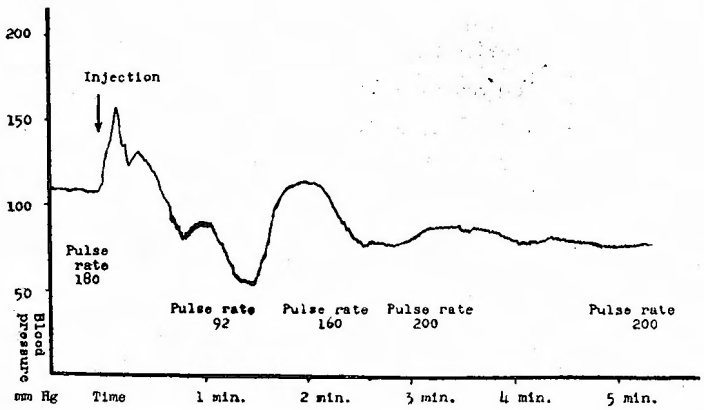

Fig. 9. Injection of 10% hydrochloric acid 5 cc into the upper abdominal cavity of cats fasting for 3 days. (No. 62)

3) The same injection in cats fasting for 7 days.

Table XIV

No.		Before injection	After injection							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
65	Blood pressure	130	198	114	112	100	96	92	96	100
	Pulse rate	220		228	212	208	220	212	216	208

4) The same injection in cats fasting for 12 days.

Table XV.

No.		Before injection	After injection							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
66	Blood pressure	110	162	126	114	110	108	102	94	100
	Pulse rate	192		201	192	188	184	180	190	192

Comment.

In No. 61, pulse rate became slow, blood pressure fell rapidly and death occurred after about 6 minutes. However, as blood pressure was considerably low before injection of hydrochloric acid, the symptoms should not be taken for those of a reflex shock, but they were merely agonal signs. Furthermore. in Nos. 63, 65 and 66 blood pressure fell gradually but not remarkably, and pulse rate showed no changes or became frequent. However, in animals Nos. 62 and 64, bradycardia and low blood pressure were observed temporarily a few minutes following injection, and soon blood pressure returned to the former level and pulse rate increased. This response is a temporary vagal reflex and not so heavy as to be called a primary shock. The typical primary shock, such as observed in the thoracic cavity, did not take place from the abdominal cavity. In short, when a severe pain was given by injection of hydrochloric acid into the abdominal cavity after fasting for about 3 days in cats, there seems to occur at first a vagal reflex and then a secondary shock-like state. However, when fasting lasts for a longer period e. g. 7 to 12 days, such a temporary vagal reflex is hardly produced.

Series V: Cooling of Body in the State of Starvation and Primary Shock.

It was observed that the shock-like state occurred more frequently about 25 per cent in winter than in summer. WAKIN and GATCH (1943) experienced that animals in shock state are liable to die in cold circumstance. I have reported in this paper that pleural shock in cats is difficult to occur in summer, but when cooled in ice-box until the rectal temperature falls to about 35.5°C, cats can be brought into shock-state even in summer. In such cooled condition of the fasting animals the following experiments were carried out.

1) Injection of 10% hydrochloric acid 5 cc into the upper abdominal cavity of cats fasting for 4 days.

Table XVI.

No.		Before injection	After injection							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
67	Blood pressure	130	170	140	130	124	122	118	110	108
	Pulse rate	192		172	172	188	176	200	200	196
68	Blood pressure	80	104	80	76	70	68	66	64	64
	Pulse rate	184		132	132	136	132	132	144	152

2) The same injection in cats fating for 7 days.

Table XVII.

No.		Before injection	After injection							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
69	Blood pressure	64	74	68	66	66	60	64	64	60
	Pulse rate	160		200	192	192	192	192	200	200

3) The same injection in cats fasting for 10 days.

Table XVIII.

No.		Before injection	After injection							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
70	Blood pressure	120	142	90	106	100	104	106	106	106
	Pulse rate	196		160	200	181	180	180	180	180

4) The same injection in cats fasting for 14 days.

Table XIX.

No.		Before injection	After injection							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
71	Blood pressure	80	132	78	84	82	80	78	80	82
	Pulse rate	164		168	144	152	160	164	164	164

Comment.

When starving cats were cooled in ice-box, rectal temperature sometimes fell suddenly, and even death occurred, and a neurogenic shock was liable to develop during the manipulation of the femoral artery for the purpose of measuring blood pressure, just as in Takeuchi's experiment, in

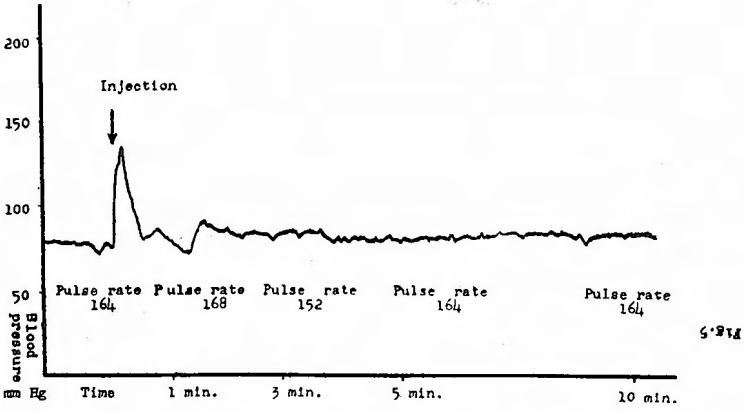

Fig. 10. Injection of 10% hydrochloric acid 5 cc into the upper abdominal cavity of cats cooled after fasting for 14 days.(No.71)

which he observed a neurogenic shock to occur immediately following the release of an arterial clamp placed on the carotid artery. The reflex shock of vagal type, however, did not occur in any case in this series of experiment. An initial vagal reflex as observed in previous Series (Ⅳ) was evoked in no case but one(No. 70).

Even in the state of instability and disorder of the autonomic nervous function due to freezing and hunger, it seems that a primary shock hardly occurs by the injection of hydrochloric acid into the upper abdominal portion of cats. If a reflex shock would be induced by stimulation of abdominal cavity in man, some additional factors, such as astonishment, fear and other emotional excitement, might play a more important rôle in its development.

Series Ⅵ : Injection of Vagostigmine in Fasting Animals and Primary Shock.

10% hydrochloric acid 5 cc was injected into the upper abdominal cavity of fasting cats about 20 to 30 minutes following the chemical stimulation of parasympathetic nerves by the administration of vagostigmine.

1) Injection of 10% hydrochloric acid 5cc into the upper abdominal cavity after preliminary injection of 0.05% vagostigmine 0.1cc per kilogram of body weight.

A) In cats fasting for 2 days.

Tablea XX. (A)

No.		Before injection	After injection							
			immediately	1′	2′	3′	4′	5′	7′	10′
72	Blood pressure	120	180	110	100	100	100	100	96	96
	Pulse rate	160		152	164	172	160	160	164	160
73	Blood pressure	120	196	128	120	102	100	104	108	114
	Pulse rate	156	80	148	144	144	148	148	160	160

B) In cats fasting for 3 days.

Table XX. (B)

No.		Before injection	After injection							
			immediately	1′	2′	3′	4′	5′	7′	10′
74	Blood pressure	150	178	148	126	140	154	144	140	136
	Pulse rate	192		88	136	176	160	172	184	212

C) In cats fasting for 5 days.

Table XX. (C)

No.		Before injection	After injection							
			immediately	1′	2′	3′	4′	5′	7′	10′
75	Blood pressure	124	174	110	100	94	100	100	100	100
	Pulse rate	200		200	196	176	176	176	176	160

76	Blood pressure	130	176	130	120	108	104	106	108	100
	Pulse rate	152		104	148	148		152	156	156
77	Blood pressure	110	154	84	100	90	90	88	86	
	Pulse rate	200		152	228	216	192	192	200	

D) In cats fasting for 6 days.

Table XX. (D)

No.		Before injection	After injection							
			immediately	1′	2′	3′	4′	5′	7′	10′
78	Blood pressure	90	120	70	66	66	66	66	64	62
	Pulse rate	184		200	200	200	200	196	196	196
79	Blood pressure	140	160	130	130	130	126	122	116	120
	Pulse rate	224		208	208	216	196	208	208	212
80	Blood pressure	100	164	114	92	84	82	80	80	78
	Pulse rate	168		104	180	180	176	168	152	160

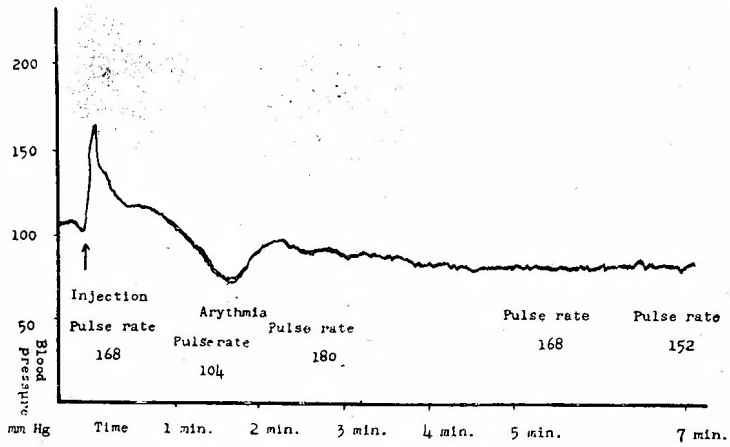

Fig. 11. Injection of 10% hydrochloric acid 5cc into the upper abdominal cavity of cats fasting for 6 days and injected preliminarily with 0.05% vagostigmine 0.1cc per kilogram of body weight. (No. 80)

E) In cats fasting for 3 weeks.

Table XX. (E)

No.		Before injection	After injection							
			immediately	1′	2′	3′	4′	5′	7′	10′
81	Blood pressure	134	192	120	122	112	110	110	112	118
	Pulse rate	208		192	172	172	168	176	176	160

A reflex shock of vagal type was not produced by injection of hydrochloric acid into the abdominal cavity following the subcutaneous injection of a small quantity of vagostigmine. However, temporary low blood pressure and bradycardia

which should be taken for a vagal reflex, was observed for a minutes following the injection of hydrochloric acid in animals Nos. 73, 74, 75 and 80.

2) Injection of 10% hydrochloric acid 5 cc into the upper abdominal cavity following preliminary injection of 0.05% vagostigmine 0.2 cc per kilogram of body weight.

A) In non-fasting cats.

Table XXI. (A)

No.		Before injection	After injection							
			immediately	1′	2′	3′	4′	4′	7′	10′
82	Blood pressure	136	166	76	70	66	62	52	54	48
	Pulse rate	196		224	196	192	192	200	176	176
83	Blood pressure	90	124	86	86	90	80	74	94	104
	Pulse rate	160		170	172	172	160	152	164	168

B) In cats fasting for 3 day.

Table XXI. (B)

No.		Before injection	After injection								Remarks
			immediately	1′	2′	3′	4′	5′	7′	10′	
84	Blood pressure	90	110	90	80	70	70	61	42	30	Died 13′
	Pulse rate	192		180	176	160	160	160	150	80	later

C) In cats fasting for 7 days.

Table XXI. (C)

No.		Before injection	After injection						Remarks
			immediately	1′	2′	3′	4′	5′	
85	Blood pressure	150	120	90	66	44	30	20	Died 6′
	Pulse rate	180			120	80	64	54	later

D) In cats fasting for 8 days.

Table XXI. (D)

No.		Before injection	After injection							
			immediately	1′	2′	3′	4′	5′	7′	10′
86	Blood pressure	86	94	54	36	34	30	20	18	Died 10′
	Pulse rate	172			80	60	60	40	40	later
87	Blood pressure	120	160	118	102	92	86	92	90	98
	Pulse rate	160		148	148	144	140	128	112	112

In considering that a large quantity of vagostigmine exerts a toxic effect on respiratory and cardiovascular function, it seems to be the effect of vagostigmine rather than a neurogenic shock, that most of the cats in this series died

within 6 to 13 minutes after injection of hydrochloric acid.

Comment.

Even when the activity of cholinesterase is arrested by vagostigmine and therefore the vagus becomes highly sensitive, the reflex shock does not result from the injection of hydrochloric acid into the abdominal cavity, whereas a moderate vagal reflex develops temporarily. Many cats died within several minutes after injection of hydrochloric acid as a result of toxicity of vagostigmine.

Series VII : Iniection of 10% Hydrochloric Acid 5 cc into the Upper Abdominal Cavity of Cats Immediately after Food-Intake.

Table XXII.

No.		Before injection	After injection							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
88	Blood pressure	100	130	100	90	86	84	84	86	94
	Pulse rate	240		210	224	232	232	220	208	208
89	Blood pressure	98	130	80	90	100	100	104	110	110
	Pulse rate	208		180	200	200	208	200	192	200
90	Blood pressure	120	150	106	104	102	98	102	100	94
	Pulse rate	200		196	200	192	188	192	200	200

Comment.

In No. 89. bradycardia and low blood pressure occurred temporarily one to two minutes after injection, however, the changes in blood pressure and pulse rate were not so remarkable as in a primary shock. They mean only a temporary vagal reflex.

Series VIII : Injection of 10% Hydrochloric Acid into the Abdominal Cavity of Toads.

1) Experimental Technic.

15 hibernating toads were used. Following the so-called Engelman's hanging method, toads were fixed gently in spine position with the thorax opened and the heart exposed, the heart point being pulled up with a serphine and connected to a lever. Heart beat was recorded by means of a kymograph. All toads recieved no anaesthetics.

2) Result.

Abdomen of toads was opened. Affter the effects of laparotomy had disappeared, 1 cc of 10% hydrochloric acid was introduced into the upper abdomen. Immediately thereafter, they wriggled, showing pain reflex. In 3 cases there occurred tachycardia, in 7 no change, and in 5 bradycardia. In 2 cases of the last group the typical temporary cardiac arrest follo-

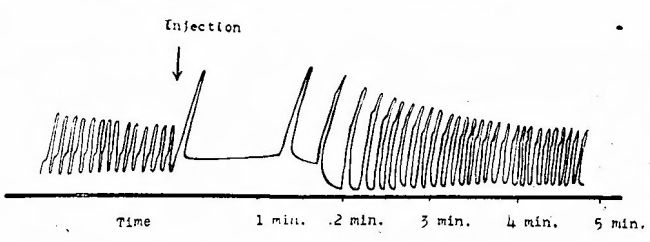

Fig. 12. Injection of 10% hydrochloric acid into the abdominal cavity of toads.

wed by marked bradycardia were observed.

3) Comment.

Even in toads the injection of hydrochloric acid into the abdominal cavity caused a marked bradycardia rather infrequently. Such bradycardia was observed only in so-called vagotonic animals and tended to be preceded by a temporary cardiac arrest. In this series heart beat of toads returned to preinjection state after about 2 minutes. In considering that in the experiment of pleural shock of cats, the decrease of pulse rate was parallel to the lowering of blood pressure, the latter change seems to have taken place also in toads. Such changes may be taken for a primary shock-like response.

DISCUSSION

Both in the experiment in rabbits by TAKEUCHI and that in cats by me, the primary shock showing bradycardia and low blood pressure was not produced by the stimulation of the upper abdomen, where parasympathetic nerve seems to be predominant. However, we have occasionally experienced in man a vagal reflex effecting temporary low blood pressure and bradycardia in surgical operations on upper abdominal viscera, such as stomach, bile duct, and pancreas. A reflex phenomenon of this sort is observed not infrequently at the time of traction of the stomach or mechanical stimulation on the common bile duct by the dissection of adhesion, or even at the moment of opening the peritoneum. Clinical manifestations of the reflex are; patients complain of unpleasant sensation in the upper abdominal portion, and become pale, sweaty, but consciousness is maintained. Shortly after cessation of the stimulation, the blood pressure gradually increases and pulse rate returns to normal. Such a response is usually called "traction reflex"; and should not be called "shock". It is also evoked in the state of light anesthesia or in the initial stage of gastric ulcer perforation. The reflex took place in this study following injection of hydrochloric acid into the upper abdominal cavity of cats fasting for 2 or 3 days, but it was not so serious as to be called a neurogenic shock. However, the fact that the injection of hydrochloric acid into the abdominal cavity of toads brought about a marked bradycardia, especially in toads in vagotonic state, should indicate that the instability of autonomic nervous system would be an important factor of a primary shock in human being.

In the experiments with dogs and cats, MARTIN and BURSTEIN (1942) reported that the heart of the animals was arrested by electrical stimulation of the thoracic vagi, while the same stimulation of the abdominal vagi caused lowering of blood pressure. And many cases have been reported of sudden death due to cardiovascular and respiratory disorders during various manipulation of the vagus and its branches in the neck and thorax. In consideration of the fact, that the primary shock, which is produced in rabbits and cats by the stimulation of pleural cavity, is undoubtedly a vagal response, a primary shock of this sort seems to take place by the vagal stimulation 1) in the neck most frequently, 2) in the thorax relatively frequently, and 3) in the abdomen least frequently.

SUMMARY

1) The primary shock, in which low blood pressure and bradycardia are characteristic, did not result from the injection of irritative chemicals, such as 5% tincture of iodine or 10% hydrochloric acid, into the abdominal cavity of normal cats, whereas the secondary shock, in which low blood pressure and tachycardia are characteristic, was liable to occur.

2) The primary shock did not develop by the injection of hydrochloric acid into the abdominal cavity even in the cats with an irreversible damage of the liver caused by carbon tetrachloride or by ligation of common bile duct. Therefore the disturbance of hepatic function does not seem to be an important predisposing factor of the primary shock.

3) Similarly, following division of bilateral abdominal sympathetics, the primary shock was not evoked by the injection of 10% hydrochloric acid (5 cc) into the abdominal cavity. Only in one case the injection of hydrochloric acid caused a temporary response of bradycardia with low blood pressure.

4) Immediately after the injection of hydrochloric acid into the abdominal cavity of cats fasting for 2 to 3 days, initial low blood pressure was induced and then the secondary shock followed. However, this initial response should not be taken for a primary shock, although it may be surely a vagal reflex. And such a vagal reflex is difficult to occur in cats fasting for a longer period (7 to 12 days). Therefore it appears that fasting for 2 to 3 days may provide an adequate predisposition to evoke a vagal reflex. Taking the primary shock for a serious manifestation of a vagal reflex, the primary shock in human being would be induced by the stimulation of the abdomen in a certain predisposed condition.

5) The shock of vagal type did not develop also in the cats which were cooled after fasting.

6) Immediately following injection of hydrochloric acid into the abdominal cavity of the fasting cats in vagotonic state induced by injection of vagostigmine, a temporary vagal reflex was observed, but it was not so serious as to be called a primary shock.

7) Injection of hydrochloric acid into the abdominal cavity of hibernating toads gave rise to a vagal reflex effecting the inhibition of cardiac function, sometimes the arrest of the heart, especially in those toads in which a parasympaticotonic state was induced by a proper preliminary treatment.

8) If the primary shock would occur in man by the stimulation of abdominal viscera, it appears, from the experimental evidences in cats and others, that besides predisposing conditions above mentioned some other factors such as psychic excitement would be necessary to aggravate the instability of the autonomic nervous system. Also the cerebral anemia might serve as a supplementary factor.

III. REPEATED STROKES ON CAT'S ABDOMEN

Goltz reported that repeated strokes on abdomen of frogs caused reflex inhibition of the heart througn the vagus nerve, i.e. heart beat became slow or even

stopped. And also, it is known in Japan that a similar state occurs by an intense stroke on "MIZUOCHI" (epigastrium). Following experiments were carried out to examine, whether a primary shock-like response results from the strokes on abdomen of cats. Cats were fixed tightly in spine position without anesthesia, and repeated strokes on the upper abdominal wall were given from 30 to 50 times in about 15 seconds.

Table XXIII.

No.		Before strokes	After strokes						
			immed-iately	1′	2′	3′	4′	5′	7′
91	Blood pressure	130	130	140	120	126	120	124	128
	Pulse rate	188		216	200	208	200	200	200
92	Blood pressure	110	86	100	86	90	94	96	100
	Pulse rate	220		240	220	218	200	200	200
93	Blood pressure	110	140	144	108	91	94	96	96
	Pulse rate	200		120	188	188	188	182	182

In No. 91, blood pressure and pulse rate showed no marked change. In No. 92, blood pressure dropped markedly immediately following cessation of strokes, but soon returned to an almost normal level. Pulse rate became rather frequent. In No. 93, pulse rate decresaed immediately after strokes, but blood pressure was raised. In summary typical low blood pressure and bradycardia occurred in no case by stroking the abdomen of normal cats.

Next, the same test was made under local anesthesia of the abdominal wall with 0.5% procaine solution.

Table XXIV

No.		Before strokes	Ater strokes						
			immed-iately	1′	2′	3′	4′	5′	7′
94	Blood pressure	126	104	76	72	70	74	76	76
	Pulse rate	118		212	200	188	176	176	180
95	Blood pressure	104	66	64	68	68	70	70	74
	Pulse rate	180		192	180	180	180	180	180

As soon as the stroking ceased, blood pressure fell pronouncedly, but pulse rate rather increased. Namely, bradycardia with low blood pressure did not ensue.

Tapping test of Goltz was reexamined with 15 hibernating toads. A typical arrest of the heart or a marked bradycardia occurred in only one case, but moderate bradycardia responses resulted frequently from stroking.

Then, the stroking experiments were carried out in cats anesthetized with urethane.

Series I: Stroking of the Abdominal wall of cats between Meals.

A) Stroking of the upper abdominal wall.

Table XXV. (A)

No.		Before strokes	After strokes						
			immed-iately	1′	2′	3′	4′	5′	7′
96	Blood pressure	120	90	108	100	88	90	100	110
	Pulse rate	172	168	160	160	152	152	148	156
97	Blood pressure	114	84	86	94	104	108	110	114
	Pulse rate	144	136	124	124	144	144	144	144
98	Blood pressure	120	88	103	104	107	117	120	
	Pulse rate	200	200	200	200	200	204	204	
99	Blood pressure	106	58	63	63	66	56	50	66
	Pulse rate	176	160	160	168	160	160	160	160

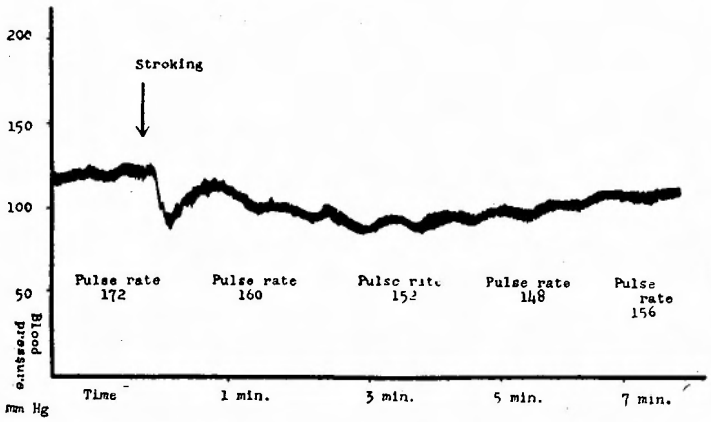

Fig. 13. Stroking of the upper abdominal wall. (No. 96)

In all cases, blood pressure fell pronouncedly following cessation of strokes and pulse rate somewhat decreased.

B) Stroking of the middle abdominal wall.

Table XXV. (B)

No.		Before strokes	After strokes						
			immed-iately	1′	2′	3′	4′	5′	7′
96	Blood pressure	126	116	106	111	118	118	118	118
	Pulse rate	160		140	156	156	156	160	160
97	Blood pressure	116	96	100	96	98	100	100	
	Pulse rate	140	140	136	136	140	140	140	
98	Blood pressure	120	112	117	120	120	120	120	
	Pulse rate	216		216	200	200	200	200	

Blood pressure fell a little, but pulse rate showed no marked change.

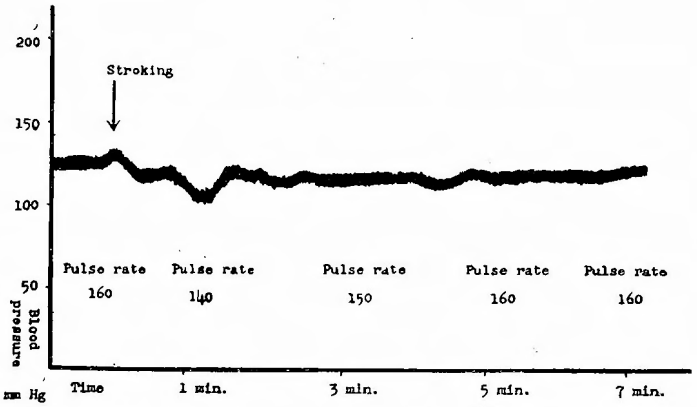

Fig. 14. Stroking of the middle abdominal wall. (No. 96)

C) Stroking of the lower abdominal wall.

Table XXV. (C)

No.		Before strokes	After strokes						
			immed-iately	1′	2′	3′	4′	5′	7′
96	Blood pressure	120	110	126	120	120	122	120	122
	Pulse rate	160		168	168	164	160	160	160
97	Blood pressure	116	118	118	116	118	116	118	
	Pulse rate	144	132	136	136	136	136	136	
98	Blood pressure	120	118	120	125	126	126	126	
	Pulse rate	200	200	220	216	216	216	216	
99	Blood pressure	100	98	99	106	110	110	112	114
	Pulse rate	176	160	164	160	172	170	176	176

Blood pressure was raised a little and pulse rate became rather frequent.

Comment.

Low blood pressure responses were evoked markedly by stroking of the abdominal wall in its upper part, moderately in the middle part, and nearly none in the lower part. Pulse rate decreased a little in stroking of the upper abdominal wall, but in Series 1 (B) and (C), it showed no change or became rather frequent. Thus, decrease in both blood pressure and pulse rate was obtained by the strokes on the upper abdominal wall only, in which the parasy-

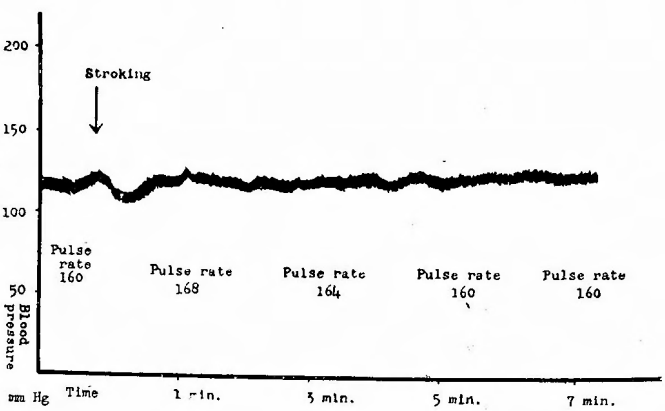

Fig. 15. Stroking of the lower abdominal wall. (No. 96)

mpathetics are predominant. However, it was not so serious as to be called a shock.

Series II : Stroking of the Upper Abdominal Wall after Food-Intake.

Tnble XXVI.

No.		Before strokes	After strokes						
			immed-iately	1′	2′	3′	4′	5′	7′
100	Blood pressure	126	92	56	50	96	108	116	116
	Pulse rate	240	220	220	120	220	220	220	224
101	Blood pressure	102	63	80	61	49	46	52	61
	Pulse rate	216		200	208	200	200	183	188
102	Blood pressure	100	44	54	46	28	40	40	36
	Pulse rate	160		100	140	160	140	110	160

In all cases, pulse rate more or less decreased. Nausea followed by a remarkable bradycardia was noticed in Nos. 100 and 102, and arhythmia for 3 minutes in No. 102. Blood pressure fell pronouncedly in all cases for 10 minutes or more.

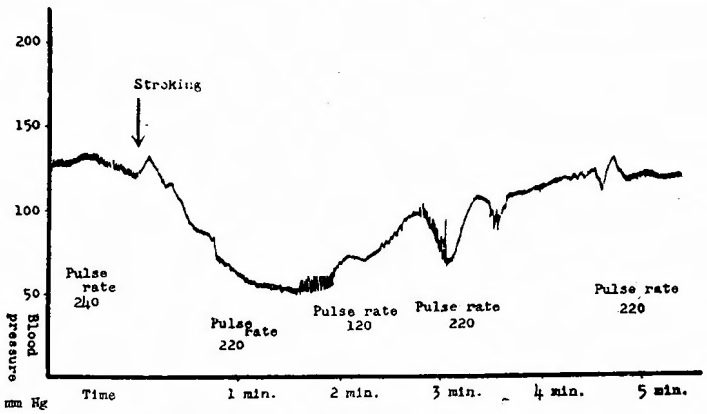

Fig. 16. Stroking of the upper abdominal wall after food-intake. (No. 100)

Comment.

In this series of cats, in which the stomach was filled with food, remarkable lowering of blood pressure and slight decrease in pulse rate were observed. Nausea and arhythmia were noticed in some cases. Thus, a cardiovascular response effecting low blood pressure and slow pulse rate was evoked in cats after feeding and under anesthesia with urethane. Though it was not such a typical bradycardia as seen in pleural stimulation in cats or in tapping of the abdomen in toads, it may be called a primary shock-like symptom.

Series III : Stroking of the Upper Abdominal wall Following Sectioning of Bilateral Vagal Nerves.

Following bilateral division of vagi in the neck dyspnoea appeared and tracheotomy was necessary. Then the cats were stroked on the upper abdomen.

Comment.

Blood pressure fell pronouncedly, but pulse rate rather increased. Thus it was shown that bradycardia did not result after division of bilateral vagal nerves.

Table XXVII.

No.		Before strokes	After strokes							
			immed-iately	1′	2′	3′	4′	5′	7′	10′
103	Blood pressure	94	64	68	70	74	76	76	76	76
	Pulse rate	192		200	208	200	192	192	192	192
104	Blood pressure	102	74	76	68	64	66	68	70	70
	Pulse rate	200	200	208	208	212	216	208	200	200
105	Blood pressure	144	130	126	120	100	90	90	90	98
	Pulse rate	200		208	192	184	176	172	168	171

SUMMARY

(1) No marked change was obtained in both blood pressure and pulse rate by repeated strokes on the abdominal wall of cats without anesthesia.

(2) Giving repeated strokes on the abdominal wall under local anesthesia with 0.5% procaine solution, blood pressure fell pronouncedly, and pulse rate became rather frequent.

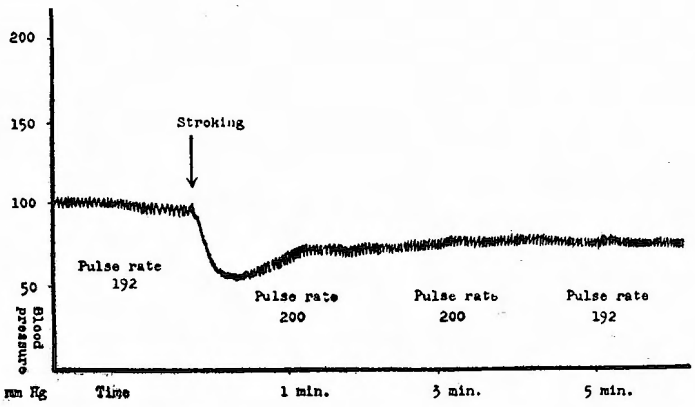

Fig 17. Stroking of the upper abdominal wall after section of bilateral vagal nerves. (No. 103)

(3) Cats anesthetized with urethane responded with bradycardia and low blood pressure most pronouncedly to repeated strokes on the upper abdominal wall, moderately to that on the middle addominal wall, but nearly none to that on the lower abdominal wall. This response seems to be a temporary vafal reflex.

(4) Decrease in both pulse rate and blood pressure was produced in cats by stroking of the upper abdominal wall immediately after food-intake under anesthesia with urethane. However, it was not such a typical bradycardia as seen in pleural shock of cats or in a critical state of toads following tapping of the abdominal wall or injecting of 10% hydrochloric acid into the abdominal cavity. It should be called a primary shock-like symptom.

(5) If the same experiment was done after bilateral sectioning of vagal nerves in the neck, blood pressure fell remarkably but pulse rate rather increased. Thus, sectioning of bilateral vagal nerves proved to inhibit the bradycardia response.

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和文抄録

神経反射性ショックの実験的研究

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徐脈，血圧下降を呈する一次性ショックを猫の胸部及び腹部に於て検討した処，次の結論を得た．

I. 肋膜性ショックの実験的研究

1) 肋膜性ショックは胸腔内に熱湯及び冷水を注入しても起し得ないが，沃度チンキ及び塩酸の注入により之を起し得た．

2) 同様に，胸腔内にアセチールコリンを注入することにより肋膜性ショックを起し得たが，アドレナリン及びピロカルピンの注入によりては起し得なかつた．

3) 夏期時には肋膜性ショックは発生し難いが，氷片にて体温を下降せしめると肋膜性ショックは発生し易くなる．即ち，寒冷刺戟は肋膜性ショック準備状態と云い得る．

4) 肋膜性ショックはアトロピンの術前注射又は頸部に於ける両側迷走神経切断により阻止される処から，迷走神経は肋膜性ショックの主役を演じるものであろう．

5) かゝる肋膜性ショックは荒木教授による頭部外傷後発生する一次性ショックの型と類似し，且つ，徐脈，血圧下降型を呈する．

II. 腹部に於ける神経反射性ショックの実験的研究

1) 正常猫の腹腔内に5%沃度チンキ及び10%塩酸を注入しても一次性ショックはみられず，寧ろ頻脈，血圧下降を呈する二次性ショックが生ずる．

2) 更に術前四塩化炭素の投与及び総輸胆管の結紮

により肝機能障碍を生ぜしめた後，或は両側腹部交感神経を切除した後，腹腔内に塩酸を注入しても一次性ショックは生じない．

3) 絶食2～3日後，腹腔内に塩酸を注入すれば，注入直後，徐脈，血圧下降を示し，やがて二次性ショック状態に移行する．この注入直後の変化は一時的に迷走神経反射現象が現われたものではあるが，一次性ショックと呼ぶ程のものではない．しかして，この現象は絶食期間が7～12日の長期に亘れば起り難いところから，2～3日間の絶食時には自律神経の不安状態が高いのであろう．即ち，迷走神経性反射現象には絶食2～3日間の自律神経不安状態が大いに関係する．かゝる迷走神経反射の強く起つた場合が一次性ショックであるとすれば，以上のことから，人間に於ける腹腔よりの一次性ショックは自律神経不安状態に於て起り易いであろうことが想像される．

4) 飢餓と寒冷の条件下に於て，猫の腹腔内に塩酸を注入した実験では，徐脈，血圧下降型のショックは起らなかつた．

5) 飢餓時にワゴスチグミンを注射して，副交感神経を興奮せしめた後，塩酸を腹腔内に注入した処，注入直後，一時的の迷走神経反射現象を示したが一次性ショックという程のものではなかつた．

6) 冬眠中の蟇に於て，腹腔内塩酸注入試験を行つた処，心搏停止，徐脈を呈する迷走神経性反射現象が起つた．しかして，特に副交感神経の興奮状態の強い蟇に於て，かゝる反応が著明であつた．

7) 人に於て，腹部から一次性ショックが起り得るとすれば，それは自律神経不安定の状態の上に，更に他の因子，例へば，精神的興奮が高調し，それに疼痛及び臓器の不快感が刺戟となり，脳血行が障碍せられて脳貧血の状態となり，一次性ショックを惹起するのではなかろうか．

Ⅲ. 腹壁叩打試験

1) 無麻酔時に於ける猫の腹壁を強打しても血圧，脈搏には変化を生じなかつた．

2) プロカインで局所麻酔された猫の腹壁を強打すれば血圧は著明に下降したが，脈搏はむしろ増加した．

3) ウレタンで麻酔された猫の腹壁を強打すれば徐脈及び血圧反応は上腹部に於て強く，中腹部に於て中等度，下腹部に於ては殆んど認められなかつた．即ち，この反応は一時的の迷走神経反応であろうと思われる．

4) 摂食後，麻酔猫の腹部を強打すれば血圧，脈搏の減少を来したが，猫の肋膜性ショックや，蟇の腹壁叩打及び腹腔内塩酸注入によりみられた定型的な徐脈は得られなかつた．即ち，これは一次性ショック類似の症状であろう．

脾臓機能亢進症に於ける脾臓

The Spleen in Hypersplenism

R. J. Leffer

Am. J. Pathol. vol. 28 p. 303, 1952

脾臓機能亢進症，即ちバンティー氏病，先天性溶血性黄疸，特発性血小板減少性紫斑病，更に最近では脾性白血球減少症，脾性貧血等に於て，脾臓は重要な役割をしめるものであると考えられて来たが，先天性溶血性黄疸を除いては，殆んどこれらの組織病理学的変化は明らかにされていない．私は，末梢血球系減少，骨髄増殖，スプレノメガリー，脾摘出術後の貧血恢復等の臨床的特色を示す脾臓機能亢進症84例と，対照例221例の脾組織を顕微鏡的に検した結果，脾摘出術前にレントゲン治療をうけた1例のバンティー氏病を除く脾臓機能亢進症例のことごとくに於て，マルピギー氏小体周縁層の巨大なことを発見した．同様な変化は224対照例中13例即ち5.8%に於てもみとめられたのであるが，脾臓機能亢進症例のそれ程明確ではなかつた．脾臓マルピギー小体の巨大な周縁層は，脾臓機能亢進症の診断的特色として価値のあるものかも知れない．

(戸部隆吉抄訳)